

CONTRIBUTION TO THE SYMPTOMATOLOGY OF INTRACRANIAL DISEASE.*

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Modern neurology is justly proud of the achievements in the recognition and management of intracranial affections, especially brain abscesses and tumors. But we must all concur in the opinion of Oppenheim, expressed at the late International Medical Congress at Moscow, that the diagnosis of these conditions is yet far from what it should be, and that the uncertainty of diagnosis is the cause of therapeutical, especially surgical, failures. So I venture to add to the abundance of clinical reports the history of two cases, if only for the sake of fortifying and supporting facts already known and established. The cases that I am about to report have given me much clinical and therapeutical information, and I therefore ask indulgence through the lengthy recital.

Case I. The patient, a boy seven and a half years old, was brought to the Montefiore Home, September 2, 1896, by his mother. She related in a husky voice and hurried language the history of her son's illness. The explanation of the mother's unusual behavior was readily apparent in her tremor, struma, and exophthalmus. She said that she was the only member of her family who was not well. Repeated interrogation failed to elicit the statement of disease of any kind in the little patient's ancestry, except the condition of the mother. The child was born at full term, grew and developed normally, had croup and measles in infancy. He attended school, and, so far as could be learned, was bright and normal in every respect until April, 1896. At this time, one day while at dinner he suddenly took the spoon with which he was feeding himself and shifted it from the right to the left hand and continued his meal. When asked the reason, he simply stated that he was unable to do it with the right. Soon afterward the mother noticed clumsiness of the right lower extremity, and the boy frequently asked to have removed from his shoe a nail which, he thought, impeded locomotion.

* This paper was read, and one of the patients and the specimen from the other were presented before the New York Neurological Society, January, 1898.

These symptoms continued for about three weeks; and after a stay of about two weeks at the Post-Graduate Hospital, the patient left considerably improved.

Two months before coming to the Montefiore Home, attacks of vomiting occurred and the patient complained of severe headache, and became dull and drowsy. The motor impairment of the right side grew worse, and a right facial palsy developed.

The mother claimed to have noticed twitchings, "grasping-like movements," of the left upper extremity, especially when the patient was asleep, and she stated further that he moved the right upper extremity when asleep in a way that he was unable to do when awake.

At no time was there any disturbance of speech noticed by the mother, and there was no incontinence of the bladder or rectum.

The mother was entirely ignorant of any cause for the onset of these symptoms, such as traumatism or acute febrile disease, and she did not notice any minor premonitions of the symptoms.

Examination. The patient was fairly well built, and weighed 41 pounds. The lower abdomen was somewhat protruding. The body showed a number of bruises, which were the results of frequent falls. The lower extremities showed a few pigmented, atrophic, cutaneous scars. Glands, especially in the groin and cervical region, were small, round, and hard. Head, asymmetrical, left parietal bone somewhat more highly arched. There was a slight convergent squint, and the ears were large. The incisor teeth were somewhat serrated, although they did not approach the so-called Hutchinson type, for they showed a convexity, not a concavity, in the middle, and the enamel was intact. Right angle of the mouth drooped. Right upper extremity was rotated outward at the shoulder, semiflexed and pronated at the elbow; fingers flexed at the second phalangeal joint, the thumb abducted. The right foot was incurvated. When the patient stood, the eyes open, there was distinct swaying, which increased when the eyes were closed, as did also the play of the tendons of the right foot (fork symptom). The gait may best be described as ataxic-hemiplegic. The deviation from a pure hemiplegic gait was that the patient put his right foot to the floor with varying force at different times, and frequently on top of the other foot.

Pulse 72, soft and small. Respiration 24; temperature 99°. Percussion of thorax showed nothing abnormal, but the note on the right side was of higher pitch than on the left. Respiratory murmurs; inspiration, as well as expiration, diminished on the right side. Heart dulness was a trifle increased to left side. The rhythm was slow, the sounds muffled.

The lower abdomen was bulging, and on palpation it conveyed to the finger the sensation of a cystic mass, situated in the middle line. This tumefaction was taken for a distended bladder, and patient was given a vessel and asked to pass urine. It took him more than fifteen minutes to start the stream, and he then passed about eight ounces of urine, whereupon the tumefaction disappeared. The urine was pale, of neutral reaction, specific gravity 1003, without abnormal constituents.

Nervous system. The patient showed considerable hebetude, although he answered questions correctly and did everything that he was told to do, but rather slowly, after long deliberation and in an automatic fashion. One could elicit no distinct defects of the intellectual or associative faculties, and there were especially no aphasic symptoms. Speech was slow and stammering. He wrote *very* poorly with the right hand, the letters being ataxic; and he did not write much better with the left hand.

Vision seemed somewhat impaired on both sides; there was no hemianopsia. Optic discs were not very clearly outlined, and there was distinct tumefaction of the right disc. The pupils were of medium size, unequal, the right wider. The reactions were prompt and alike on both sides. Although each eyeball could be moved freely in every direction, the conjugated excursions of the eyeballs were insufficiently balanced and accompanied by irregular rotatory and lateral nystagmus-like movements. The muscles mostly affected seemed to be the left external and right internal recti.

Smell and taste were unimpaired; hearing was distinctly impaired on the right side; the bone conduction on this side was absent. Both drum membranes were retracted, especially the right.

It was seen at a glance that the patient had an inequality of facial innervation. This was conspicuous when the countenance was in repose, and when it displayed spontaneous, unconjured emotions. On intentional and volitional innervation the two sides of the face responded equally. When the patient was told to laugh, *i. e.*, to make facial grimaces, the external equivalent of laughing, the asymmetry of the face was not seen, but at once became evident when the laugh was the externalization of a psychological equivalent. Moreover, it was to be remarked that the facial asymmetry was more conspicuous when the patient smiled than when he laughed aloud. If the smile was preparatory to hearty laughter, the unequal innervation of the face was first very manifest, and as the smile transformed into laughter it gradually became less evident and finally dis-

appeared, while the laughter continued in a spasmodic way for some time.

Innervation of the soft palate was equal on both sides, but the tongue apparently deviated to the right.

Percussion of the skull was not painful, and auscultation of the percussion did not yield any information.

The right upper extremity was flabby and flaccid, of smaller volume than the left, and passive movements encountered no resistance.

Right lower extremity showed similar abnormality; the large toe was in a state of hyperextension, and the foot showed considerable excavation of the plantar surface.

Motor power of the right upper extremity was diminished, and this was more evident when the co-ordinating functions were examined than by dynamometric examination. There was no tremor of the upper extremity.

The motor power of right lower extremity was hardly diminished; but it was very difficult for the patient to move the toes or right ankle joint properly and to the normal extent. Very distinct ataxia in the right lower extremity.

Electrical reactions of the muscles of the right upper and lower extremities were diminished only to a slight degree.

The superficial reflexes were lessened on the right side. Patellar jerk livelier on right side, and there was an easily exhaustible ankle-clonus on this side. Jaw-jerk was present.

Examination of sensibility was rather unsatisfactory, on account of the age and mental condition of the patient; but positively there was no disturbance of sensibility of any account.

The patient was seen by Dr. Dana in the afternoon of the day when this examination was made, and the diagnosis of a subcortical brain tumor was confirmed. It seemed justifiable to locate the lesion in or near the optic thalamus.

The next morning the patient complained of severe headache and vomited frequently. He was irritable and dull, showed distinct spasmodic laughter and crying, with ready transition from one state to the other. He had not passed urine for 24 hours, and the bowels were constipated. Temperature, taken in the right axilla, was 99°; in the left axilla, 98.2. Pulse, 60, slightly irregular; respiration 18, irregular.

The state of spasticity and of the reflexes of the right extremities was rather interesting. At one moment, especially when the patient's attention was not directed towards the extremities, or when one happened to strike a moment of complete cessation of innervation, the extremities were absolutely flaccid and no reflexes could be obtained. At another moment a very slight resistance to passive movements was encountered; patellar jerk was fairly alive, and an ankle-clonus was easily demonstrated.

The patient was put on mercurial inunctions (60 grains daily), and on increasing doses of iodide of potassium. Very soon afterward there was improvement; the headache became less frequent and less intense, and he vomited only rarely. He was more wakeful, active, and playful. The dexterity of the right extremities returned to a considerable extent; the optic discs became normal on both sides, and the ocular excursions were fuller and more steady, and there was only a slight insufficiency of the conjugated excursions towards the left side. After a further lapse of about three weeks all subjective, and most of the objective, symptoms disappeared.

When examined five months later the patient exhibited the following symptoms: Right angle of mouth slightly lower. There was some swaying with eyes closed; in walking the patient carried the right upper extremity slightly flexed and rotated inwardly. Right lower extremity was held rigid at knee-joint, and from time to time the patient stumbled with right toes. Patient had forgotten what he knew about numbers and what he learned in school; in fact, he was silly, laughed spasmodically on the slightest provocation, and did it so persistently that his teacher was obliged to dismiss him from school. The skull was slightly tender to percussion over the left parietal region. There was convergent strabismus; left internus was in a state of slight contracture. Pupillary reactions were perfect. When the patient was asked to follow the examiner's fingers with his eyes there was noticed:

1. A decided sluggishness of execution, the eyeballs moving very slowly towards the direction indicated.
2. When in lateral position a deviation of the eyeball in the vertical direction; for instance, when looking towards the right an overaction of the superior oblique, and towards the left an overaction of the inferior oblique occurred.
3. All ocular movements were accompanied by nystagmus-like movements.

When the head was not supported it drooped slightly. To voluntary innervation right and left facial nerve responded quite normally, but on talking, or in repose, or on emotional display, a paralysis of the lower branches of the right facial was evident with all the above mentioned specifications.

The right extremities were thin and flabby; the spasticity varying; disturbances of co-ordination were well marked; no sensory disturbances.

Function of bowels and bladder normal.

Case 2. M. B., man, 29 years old, a peddler, single, entered the Montefiore Home, August 20, 1895, on account of pulmonary tuberculosis. The patient was born of healthy parents, and had a good family history. In 1893 he had typhoid

fever; in 1894, while in South Africa, protracted diarrhea and fever kept him in bed for two months. He was temperate, a hard-working man, free from venereal infections. He considered himself healthy until the symptoms of the present disease appeared about three years ago.

The usual forerunners of tuberculous infection were soon followed by pyrosis, distress after eating, and edema of the legs. On examination the patient exhibited the symptoms of a moderate lesion of both apices, and indications of mild Bright's disease. He remained comparatively well until November, 1896, when a new set of symptoms developed.

Without any premonitory symptoms or obvious cause for the superadded illness, the patient suddenly experienced pain and paresthesias in the left half of the face, including the oral and nasal mucous membranes and the teeth on the left side. At the time of the examination he complained of slight cough; swelling of the legs; paresthesias on the left side of face; attacks of severe occipital headache, associated with dizziness and vomiting; uncertainty of locomotion, especially while turning around. He felt all the time like a drunken man; bit his tongue when eating or talking, and thought that his eyesight, especially of the left side, had become poorer.

Examination. Pulse 96°; resp. 24; temp. 97°. Urine: pale, acid; specific gravity, 1014; contained a considerable amount of albumin, hyaline and granular casts.

Patient was of large frame, but anemic, and rather poorly nourished. Edema around ankles. Abdomen and lower thoracic aperture very much distended and barrel-shaped.

Examination of the internal organs revealed lesions of both lungs. The liver was considerably enlarged, the spleen slightly; the body was covered with cold, cutaneous and periostitic abscesses in various stages of development or retrogression. The clinical picture was so complicated that I shall omit the details of the symptomatology, excepting those bearing on the subject of brain tumor.

Head: Skull asymmetrically built, was not tender to percussion, and on auscultation no abnormality was noticeable. The most prominent feature was the facial expression. The left half of the face seemed contracted. Depressions were noticed corresponding to the temporal and masseter muscles of the left side. The left eyelid drooped, and was the seat of a few small herpetic blisters. The eyeball was injected and the cornea cloudy.

On superficial inspection it appeared as if patient had a right-sided peripheral facial palsy.

Smell and taste were impaired on the left side. Vision was impaired on both sides, but optic discs appeared fairly normal.

Hearing was good, but the patient complained of subjective noises in the right ear. Pupils of medium size, well shaped, equal on both sides, and reacted promptly. Nystagmiform twitchings accompanied the ocular excursions in all directions, most marked, however, when the eyes were turned to the left or upwards.

When the patient was put through the usual tests to determine integrity of the facial nerve, the right nerve was found to be entirely normal, while in the distribution of the left a phenomenon was noticed that can best be compared to the ataxia and over-innervation frequently seen in tabic patients. The patient performed all motor functions with the left facial—closing and opening the eyes, showing the teeth, frowning, etc.—with unnecessary force, with an expenditure of energy that was extravagant in comparison with the effect desired.

He was unable to chew with the left side, and on swallowing he experienced slight difficulty with large pieces. Food frequently lodged on the left side, and he was unable to dislodge it in the usual way. The excursions of the lower jaw were limited, the patient being unable to open his mouth as wide as he used to, and when doing so the protuberantia mentalis deviated towards the right side, probably on account of weakness of the left pterygoid muscles. Temporal and masseter muscles were atrophied, their mechanical irritability was increased, and the electrical reactions markedly but only quantitatively diminished. The latter was true also of the left facial muscles.

Sensibility of face was subjectively and objectively disturbed, and the patient himself mapped out the course of the first and second branches of the trigeminal nerve by indicating the situation of the paresthesia.

The objective sensory findings are shown by the accompanying figure. The sensory disturbances included the oral and nasal mucous membranes, and the left half of the tongue and conjunctiva. The tongue was dried and furred, particularly the left half. It protruded straight, showed no tremor, and no inequality of volume. Innervation of the soft palate was normal, and motility and sensibility of the pharynx and larynx seemed undisturbed. Motility and sensibility of the upper and lower extremities were normal; the knee-jerks were very much diminished; there was slight Romberg symptom, but no evidence of ataxia, and the gait was slightly cerebellar.

The patient was ordered mercurial inunctions—75 grains daily—and increasing doses of iodide of potassium.

When examined after two weeks, the patient complained of severe left-sided headache, coming in attacks and lasting from $\frac{1}{2}$ to $1\frac{1}{2}$ hours. Vertigo and dizziness were very severe, so much so that the patient was unable to be out of bed. Appetite and bowels were fairly normal; micturition frequent.

The gait was uncertain, swaying, typically cerebellar. Romberg sign was distinct. The knee-jerks were very much diminished. Head and cranial nerves as before.

Treatment was continued for three weeks longer, and at the end of this time most of the symptoms disappeared; headache and vomiting ceased entirely, locomotion was undis-



FIG. I. *n*, area of absolute anesthesia; *o*, area of tactile anesthesia; *P*, area of paresthesia.

turbed, knee-jerks lively, sensibility of left side considerably improved.

He was seen by Dr. Sachs on two occasions, and the diagnosis of a lesion, meningitis or tumor at the base of the brain in the posterior fossa at the angle between the pons, oblongata and cerebellum was thought to be the correct one.

Unfortunately, the pulmonary and renal lesions were less amenable to treatment, and the patient finally succumbed in uremic coma; but at no other time were any brain symptoms noticed.

The post-mortem examination was made by F. W. Vissman, to whom I am indebted for the following notes:

Spinal cord showed no abnormalities; the arachnoid was

very slightly edematous in the upper cervical portions. The dura mater of the brain showed no abnormality; the arachnoid was slightly edematous over the vertex, and at the base it contained a number of gray, white nodules smaller than a pinhead. The lateral ventricles were of normal size and content. The ependyma was smooth, glistening and transparent. The vessels at base were thin and free from disease.

The pericardium contained about two ounces of a clear, straw-colored fluid; the walls of the sac were smooth and glistening, and there were no thickened spots. The heart itself was about the size of the fist of the corpse; the muscle structure was very flabby, and of a grayish-reddish color. The valves were soft and pliable, not thickened.

Parietal and visceral pleuræ were firmly adherent to one another over nearly the entire surface of the lung. On dissecting the apex of the size of a pigeon's egg was found, containing a creamy white liquid. There were a number of other cavities in the lungs, which varied in size from those described down to those of the size of a cherry. The walls of these cavities were rather clean. In other portions of the lung we found the air cells filled with dark, red blood, which gave the cut surface a rough, velvety appearance. In other places the air cells were dilated, but contained air. The right lung showed practically the same changes as the left lung, excepting that it contained no cavity with pus.

The spleen was enlarged and very firm; its color was dark red; on the cut surface numerous glassy-looking spots about the size of a hempseed were visible. Capsules smooth and transparent.

The left kidney was large and grayish-yellow in color. Its surface was very slightly mottled and smooth. The cut surface showed the cortical portion thick and very slightly prominent above the medullary portions, and very pale. In right kidney the same conditions were found as in the left.

The liver was large, the edges round, the color was grayish-red, consistency very firm. The cut surface appeared cloudy. The central veins and the lobule were not easily distinguished with the naked eye.

Immediately below the left kidney a tumor of the size and shape of a goose-egg was found. This tumor seemed to be attached by a pedicle to the left side of the bodies of the fourth and fifth lumbar vertebræ. From there it extended down about $2\frac{1}{2}$ to 3 inches in the psoas muscle. On removing the tumor it was accidentally cut and a creamy, white fluid flowed out. The inner surface of the cyst was smooth, except where it emanated from the last lumbar vertebra: there was a rough erosion, which admitted the end of the little finger.

Diagnosis: Meningitis basilaris tuberculosa; Pleuritis adhæsiva chron.; Phthisis pulmonum; Hydrops pericardi; Hypertrophia lienis; Degeneratio-amyloidea lienis; Hepatitis parenchymatosa et interstitialis chron.; Caries ossis vertebræ lumbalis V.

I will add that there was some dispute at the post-mortem table about the existence of any intracranial lesion at all. To me the pia mater at the base of the brain in the left ponto-medullo-cerebellar angle seemed thickened, and a few white, rough streaks were seen. But my belief was not shared by the others present. On the contrary, the opinion was expressed that the whole symptom-complex was a functional one, and probably caused by the renal affection—uremic in nature.

Twenty-four hours afterwards, when the specimens were looked at again after a stay in Müller's fluid, a unanimity of opinion that the pia was abnormal was reached. In addition a few small nodules of the size of a pinhead were noticed on the specimens.

A review of the symptoms of these two cases necessitates further comment:

The diagnosis of an organic, intracranial lesion, in fact of a brain tumor, was positive in the first case, but whether the nervous symptom-complex of the second case could not just as well be explained by the assumption of a functional, say uremic toxemia, as was suggested at the autopsy table, had to be carefully considered.

Of course, the constancy and the clearness of the clinical picture, the absence of other uremic symptoms, and the presence of some undoubtedly organic symptoms, pointed rather strongly towards the organic nature of the disease, tumor or meningitis.

The positive discrimination between a solid tumor or localized basal meningitis was in this case, and is often, almost impossible. During life the absence of a stiff neck and of permanent headache were more in favor of the diagnosis tumor, and from the post-mortem findings the limited localization, the normal appearance of the rest of the meninges, seem to point in the same direction. In determining the nature of the lesion the age of the respective patients was somewhat misleading.

Following the teachings of text-books, such as Gowers', Dana's, Sachs', Starr's, Dercum's, Oppenheim's, and others, in which rarity of gummatous lesions in children

and prevalence of such in adults is strongly emphasized, one would at first feel inclined to regard the lesion in the first case as a tubercle, and to suppose a specific infection in the second. But the pulmonary affection of the second patient (although tubercle bacilli were never found in the sputum despite repeated examination); and the swollen glands, the formation of the teeth, and the cutaneous scars in the first case, equivocal as their interpretation may be, were of considerable aid in the decision. The remissions and the interrupted course of the disease in the first case were of great value in deciding in favor of a specific lesion.

The general symptoms of intracranial affections were present in both cases; in the first more marked than in the second; the absence of optic neuritis in the second case is the more remarkable, as this symptom is said to be very common in affections of the posterior fossa.

The special and localizing symptoms of the second case presented a fairly clear ensemble, characteristic of lesions of this part of the brain. Whereas, the disease of the fifth nerve made the basal and superficial position of the lesion highly probable; the purely cerebellar symptoms of dizziness, cerebellar gait, and especially diminution of the knee-jerks, were perhaps too strongly outspoken for a lesion at this situation, a lesion that presumably was not very large, as the absence of disease of other nerves of the base of the brain, and especially the absence of bulbar symptoms, seemed to indicate.

In this connection it is well to call attention to the symptom pointed out by Stieglitz as being of great value in the initial diagnosis of cerebellar lesions; I mean the diminution of the electrical response of the facial nerve, which was present in our case.

Affection of the motor portion of the trigeminal is a rather uncommon feature of trunk lesions of this nerve. Leube states that in trunk lesions of the fifth nerve the sensory fibers are frequently the only ones to suffer, the initial irritation (neuralgia) being soon followed by paralysis (anesthesia).

Concerning the lesion of the fifth nerve, especially of its motor portion, the clinical evidence of a disease of the pterygoid muscles deserves special mention. The limitation of

the excursions of the left mandibular joint and the deviation of the lower jaw towards the healthy side could hardly be explained on any other basis. The symptomatology of the lesion of the sensory branches shows in our case a feature rarely described: the above-mentioned over-innervation of the facial muscles—let us call it the facial ataxia.

And, in fact, one often notices in tabic patients an undue widening of the palpebral fissure, alternating with a slight ptosis. Every time that I have found this symptom there has been disease of the fifth nerve.

It brings forcibly to one's mind the belief that a great deal of the purely ataxic symptoms observed in other cases, especially in cases of tabes, is conditioned by an interference with sensory, or, better said, centripetal impressions.

The occurrence of trophic disturbances—herpes, injection of the eyeball, and cloudiness of the cornea—is not entirely in accord with recent physiological teaching, explaining these symptoms by the assumption of a nuclear lesion or a disease of the Gasserian ganglion.

We are not justified from the post-mortem findings in venturing an explanation of the diminished reflexes; and to speculate about it seems unwise and untimely when new theories about this subject are brought out almost daily.

The symptomatology of the first case was very striking, and, in view of the scarcity of cases of this type, seems to deserve a more detailed consideration.

Cases of tumor of the optic thalamus have been reported by Nothnagel, Dejerine, Dana, Oppenheim, Dercum, Sinkler, and others; but in most of them the lesion was rather extensive, and it is somewhat difficult to enucleate the clear picture of thalamic affection.

Before going further, it seems suitable to recapitulate briefly the anatomy and physiology of this part of the nervous system. Our knowledge in this direction is meagre indeed, but only recently a noteworthy addition has been made to it by Exner, Bechterew, Simbriger, and others. Frequently the statement is made that disease of the basal ganglia rarely gives rise to any special localizing symptoms throughout the whole course when general symptoms are fully developed, and in the last

edition of the "Traité de Médecine" (Vol VI.), for instance, one finds the statement concerning tumors of the basal ganglia: "Celles-ci ont une symptomatologie beaucoup plus confuse encore. Non seulement elles n'ont rien de pathognomonique, mais elles peuvent évoluer sans symptômes. Leurs symptômes, lorsqu'elles en ont, échappent à toute combinaison nosographique. Seuls, les symptômes des tumeurs, en général, existent."

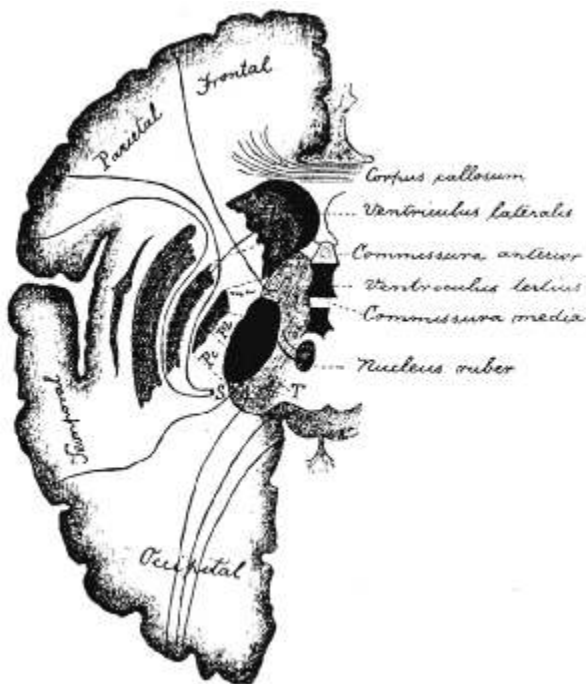


FIG. II. T, probable situation of tumor; m n, pathway for motor cranial nerves; Pb, pyramidal fibers for upper extremity; Pc, pyramidal fibers for lower extremity; carrefour sensitif.

The accompanying cut, modified after Leube, gives a clear conception of the position, surroundings, and intracranial connections of the optic thalamus that are so far known.

In a recent article in the *Neurologisches Centralblatt*, Bechterew claims to have found the centripetal and centrifugal thalamo-spinal connections. The former are supposed to be represented in fibers of the posterior columns, the latter to run

down with the pyramidal tracts, situated in the internal capsule, in front of the bundle representing these fibers here.

In the same article Bechterew calls the optic thalamus a center for the complicated psychical reflexes, a center regulating the expressions of the emotional state and the reflex functions of the vegetative organs. This author is in agreement with Exner, Starr, Dana, and other writers on the subject.

Taking it altogether, it seems that the optic thalamus is a center regulating and governing the functions midway between intellect and instinct; functions which by ancestral or personal habitual performance have become more or less permanently organized, and are generally executed without interference of consciousness.

In general, the symptomatology of the first case coincides fully with the classical descriptions characteristic of lesions of the optic thalamus. Nearly all symptoms that are mentioned in this connection were present in this case: emotional spasticity, mental hebetude, pupillary and ocular symptoms, abolition of the psychical reflex of the seventh nerve, mild and slowly progressing hemiplegia with inconstant spasticity, muscular atrophy and inco-ordination, depression of all vegetative reflexes, were and are in part present yet in this case, as in other cases hitherto reported.

The change of the percussion note of the right thorax and diminution of respiratory vigor of the viscus is a symptom that has not been described.

It is rather difficult to imagine that this occurrence is a part of the general vegetative depression, the one-sidedness being an almost insurmountable obstacle for such a conception. It would satisfy one more to assume that the change in percussion and auscultation was produced by a physical cause—the flabbiness of the resonating walls of this side. From the symptoms enumerated it is difficult to choose the ones or one that could be regarded as the direct focal symptoms.

In looking at the accompanying diagram and imagining the supposed tumor to be situated in the anterior part of the optic thalamus, the ocular and hemiplegic symptoms could be explained by pressure on the capsular pathways for the cranial motor nerves and the motor fibers for the opposite side

of the body. It would be equally well to look for an explanation of the inco-ordination in the same direction. Widening of the pupil and the lowering of temperature on the paralyzed side are frequently mentioned as thalamic symptoms, but are, unfortunately, very inconstant. The muscular atrophy, the general depression of nutrition and vegetation, are, however, no common associates of capsular hemiplegias. Uniformly the paralysis of the psychical reflex, expressed through the facial nerve, is looked upon as the characteristic thalamic symptom. The pathomechanical explanation of this symptom is entirely hypothetical. In spite of this, I would join the majority and look at the symptom as a pathognomonic one, were it not for a recent experience which makes me cautious of accepting this view unconditionally. I refer to a case of undoubted peripheral palsy of the seventh nerve that I had occasion to observe some time ago. After the improvement and disappearance of most of the symptoms, the symptom that persisted permanently was mimetic palsy. The case altogether was a rather uncommon one, inasmuch as the facial palsy was the first expression of constitutional syphilis, and it preceded the appearance of the cutaneous symptoms by several days. The roseola was to be noticed five days after the onset of the sudden facial palsy. Whereas the electrical reactions and the intentional innervation of the face muscles returned rather quickly after specific medication, the interruption of the physical reflex is still noticeable in the patient. In attempting to explain this phenomenon, bringing in the optic thalamus was manifestly out of the question. The unusual etiology in this case, being more malignant than the common cause (exposure to cold), is, perhaps, evidenced in the clinical behavior of the affection. The syphilitic poison may either have altered the structures of fibers to such an extent that they were still able to convey volitional and electrical stimuli, but not subtle psychical impulses.

It goes without saying that this argumentation does not hold good for our case. I quote this experience, not to create further confusion, but to emphasize a general clinical rule, the special application of which to our case will prove its value.

Briefly, then, this rule says that the clinical and diagnostic importance of a symptom does not depend so much upon the

symptom *per se* as upon its attributes, its character, color, tint, as it were, and its relations towards the other symptoms and to the general picture.

A symptom separated from its clinical company, or not sufficiently analyzed into its elements, loses a good deal of its value and is misleading in the final diagnostic calculations.

Now we return to our case and make application of the foregoing principle after again reviewing the symptomatology from this standpoint. The patient presented a hemiplegia, but not a hemiplegia in the usual sense of the word; a hemiplegia with very little paralysis of motor power, but pronounced paralysis of co-ordination—paralysis of a function that is either primarily instinctive or becomes so through habit. The defective ocular excursions may be looked upon in the same way as an inco-ordination of the external eye muscles. The mimetic palsy illustrates best the type of this paralysis, and lends a part of its clinical character to all the other symptoms. It is a paralysis of a purely instinctive motion, in this particular instance freed from obscuring combinations that prevent the thorough understanding and proper interpretation of the paralytic symptoms, in the eyes or extremities. The retardation of the general psychical functions, the retardation of the vesical and intestinal motility, tally very well with this conception, too.

What is, then, the pathognomonic symptom, or, better, complex of symptoms, of a localized lesion of the optic thalamus? Let us attempt to construct it.

From a review of the literature and an analysis of the case described, we would be justified in saying that a lesion of the anterior part of the optic thalamus gives rise to evidences of irritation or paralysis of the fibers situated near by, *i. e.*, the fibers of the anterior limb of the internal capsule. These symptoms are mostly or exclusively apparent on instinctive, habitual, and vegetative reflexes, the type being presented in the mimetic paralysis of the facial.

Lesion of the posterior half of the thalamus is frequently the cause of hemianopsia and other sensory disturbances. Whether an analogy could be established between the symptomatology of affections of the anterior and the posterior half of the optic thalamus future study of cases will have to decide.

Finally, let us consider the exact localization of the lesion in our case. The absence of hemianopsia or sensory disturbances on the opposite side of the body, the interference with the fibers for the cranial nerves in the capsular pathways, make it probable that the lesion was laterally in the anterior and middle portions of the optic thalamus (see figure).

The course and response to medication of our first case is a familiar occurrence. The persistence of some symptoms that are present to-day will have to be explained by the assumption that the previous lesion had destroyed some of the gray matter of the thalamus, and this destruction is like disease of gray matter in any other part of the nervous system—irreparable; or, on the other hand, by the supposition that the tumor is only arrested in its growth and somewhat shrunken.

The termination of the second case deserves a few words. The lesion was undoubtedly a tuberculous one and has, as the inspection of the specimen proves, almost entirely disappeared. The specimen further shows that no incapsulation or calcification has taken place.

With due regard to the *post hoc* fallacy, I am aware of the fact that spontaneous cures of tuberculous lesions have frequently been observed; though close relation in time between cause and effect, between specific medication and the disappearance of the clinical symptoms of the lesion, merits some consideration.